

U.S. Department of Labor

Office of Administrative Law Judges
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In the Matter of

MONA T. BRIDGES
(Widow of GEORGE H. BRIDGES)
Claimant

v.

ISLAND CREEK COAL COMPANY
Employer

v.

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS
Party in Interest

Date Issued: August 22, 2000

Case No.: 1999-BLA-775

APPEARANCES:

Mr. Frederick K. Muth, Esq.
For the Claimant

Ms. Mary Rich Maloy, Esq.
For the Employer

BEFORE:

Richard T. Stansell-Gamm
Administrative Law Judge

DECISION AND ORDER

This matter involves a claim filed by Mrs. Mona T. Bridges, widow of Mr. George H. Bridges, for survivor benefits under the Black Lung Benefits Act, Title 30, United States Code, Sections 901 to 945 ("Act"). Benefits are awarded to persons who are totally disabled within the meaning of the Act due to pneumoconiosis, or to survivors of persons who died due to pneumoconiosis. Pneumoconiosis is a dust disease of the lung arising from coal mine employment and is commonly known as black lung.

I conducted a formal hearing in Beckley, West Virginia on August 10, 1999, attended by Mrs. Bridges, Mr. Muth, and Ms. Maloy. My decision in this case is based on the testimony presented at the hearing and all the documents admitted into evidence (DX 1 to DX 25, EX 1 to EX 10, and CX 1).¹

ISSUES

1. Whether Mr. Bridges suffered from pneumoconiosis.
2. If Mr. Bridges had pneumoconiosis, whether the pneumoconiosis arose out of coal mine employment.
3. If Mr. Bridges had coal workers' pneumoconiosis, whether his death was due to pneumoconiosis.

Claimant's and Coal Miner's Backgrounds

Born on May 9, 1926, Mr. George H. Bridges married Mrs. Mona T. Bridges (Holliday) on May 18, 1951 (DX 1 and DX 7, DX 24-12, and TR, page 15). Mr. Bridges started working in coal mines in 1944 and continued his work as a coal loader, cutting machine operator, and roof bolter through October 1982 when he became sick (DX 3). From 1955 to 1971 and 1972 to October 1982, Mr. Bridges worked as a coal miner for Island Creek Coal Company (DX 2, DX 3, DX 4, and DX 5).² Unfortunately, Mr. Bridges passed away on April 1, 1984 (DX 1 and DX 8).

Procedural Background

Initial Living Miner Claim

¹The following notations appear in this decision to identify specific evidence: DX - Director exhibit, EX - Employer exhibit; CX - Claimant exhibit; ALJ - Administrative Law Judge exhibit, and TR - Transcript of hearing. At the hearing, based on the consent of both parties, I kept the record open for the submission of a medical deposition by employer's counsel. On October 4, 1999, I received the September 17, 1999 deposition of Dr. Caffrey. Absent any objection, the deposition is marked and admitted as EX 10.

²Based on Mr. Bridges' coal mine employment record (DX 2, DX 3 and DX 4) and Mrs. Bridges' hearing testimony that her husband last worked as a coal miner for Island Creek Coal Company (TR, page 16), I find Island Creek Coal Company is the responsible operator in this case. See also agent's admission of responsibility (DX 17).

Since Mr. Bridges' last coal mine employment occurred in West Virginia, the United States Court of Appeals for the Fourth Circuit has jurisdiction over this case. See *Shupe v. Director, OWCP*, 12 B.L.R. 1-200, 1-202 (1989)(en banc).

On June 28, 1982, Mr. George Bridges filed a claim for Federal black lung disability benefits with the United States Department of Labor (“DOL”).³ On October 13, 1983, while DOL was considering Mr. Bridges’ claim, the West Virginia State Occupational Pneumoconiosis Fund found Mr. Bridges to be totally disabled due to pneumoconiosis and awarded total disability benefits, with an effective date of October 14, 1982.⁴

Following a November 1983 pulmonary examination by Dr. John M. Daniel, DOL, on January 27, 1984, notified Mr. Bridges he was entitled to disability benefits, with disability occurring as of October 1, 1982.

Shortly after the favorable DOL decision, Mr. Bridges died on April 1, 1984. On May 10, 1984, an agent for Island Creek Coal Company executed an agreement to pay black lung disability payments. The agent also noted that since Mr. Bridges had received a total award by the West Virginia State Occupational Pneumoconiosis Fund as of October 14, 1982, the company expected a dollar for dollar offset. On August 17, 1984, the West Virginia State Occupational Pneumoconiosis Fund notified Mrs. Bridges that she would receive 104 weeks of benefits as the widow of Mr. Bridges. On January 18, 1985, the DOL District Director entered his final decision and order awarding benefits to Mr. Bridges from October 1, 1982 through March 31, 1984. The DOL award was then offset by the West Virginia black lung disability payments.

Withdrawn Survivor Claim

On May 18, 1984, Mrs. Mona Bridges applied with DOL Federal Black Lung disability benefits as an eligible survivor of a deceased coal miner (DX 10).⁵ DOL notified Island Creek Coal Company of Mrs. Bridges’ claim on June 4, 1984. On January 17, 1985, DOL denied the claim. Mrs. Bridges, through her representative,⁶ requested a hearing before the Office of Administrative Law Judges (“OALJ”) on February 12, 1985. DOL forwarded the claim to OALJ in March 1985.

Subsequently, Administrative Law Judge Glenn Lawrence scheduled a hearing for February 5, 1987. However, counsel for the employer requested a continuance in January 1987 due to counsel’s inability to obtain a medical release authorization from Mrs. Bridges. Mrs. Bridges’ representative did not object to the continuance. The next month, February 1987, the hearing was continued. On October 28,

³DX 24 contains portions of both Mr. Bridges’ living miner claim and Mrs. Bridges’ withdrawn survivor claim.

⁴The West Virginia State Occupational Pneumoconiosis Fund had previously awarded several partial disability payments: 25% in 1974, an additional 15% in 1980, and an additional 5% in 1983 (DX 24-5).

⁵See DX 24.

⁶During this time frame, Mrs. Bridges was represented by a member of the United Mine Workers of America Benefit Service Fund.

1987, Mrs. Bridges' representative withdrew from the case. The next day, October 29, 1987, Administrative Law Judge David Clarke conducted a formal hearing in Beckley, West Virginia. At the hearing, counsel for the employer asked for another continuance due to his inability to obtain Mr. Bridges' autopsy slides because he did not have an updated medical release authorization from Mrs. Bridges. While she did not object to the continuance, Mrs. Bridges explained she had not received any of the documents. Judge Clarke continued the hearing, noting on the record that Mrs. Bridges had signed before him a document authorizing the release of Mr. Bridges' autopsy slides. In January 1988, Administrative Law Judge Robert Amery notified Mrs. Bridges of his intention to conduct a hearing on March 15, 1988. In response, Mrs. Bridges withdrew her request for a hearing because she had been receiving benefits from the state of West Virginia for two years and understood that she could not also receive Federal survivor benefits.⁷ Accordingly, Judge Amery remanded the claim to the DOL District Director.

After Mrs. Bridges executed a formal DOL document requesting withdrawal of her claim, which indicated she retained the right to file the Federal survivor claim in the future, the District Director approved the withdrawal of her survivor claim on June 6, 1988.

Present Survivor Claim

On May 18, 1998, Mrs. Bridges renewed her claim with DOL for survivor benefits (DX 1). After a medical review of Mr. Bridges' medical record, death certificate and autopsy report (DX 12), DOL, in early Spring 1999, notified Island Creek Coal Company of its finding that Mrs. Bridges was entitled to survivor benefits (DX 18 and DX 21). On February 25, 1999, Island Creek Coal Company contested her entitlement to survivor benefits and requested a hearing with OALJ (DX 19 and 22). On March 2, 1999, DOL initiated interim survivor benefits, effective February 2, 1999 at the rate of \$469.50 per month (DX 23). DOL then forwarded the case to OALJ on April 7, 1999 (DX 25). Pursuant to a Notice of Hearing, dated June 11, 1999, I conducted a hearing on August 10, 1999.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Stipulations of Fact

At the hearing, the parties stipulated to the following facts: (1) Mr. George Bridges was a coal miner with post-1969 coal mine employment; and, (2) Mr. Bridges had at least 26 years of coal mine employment (TR, pages 7 and 8).

Elements of Entitlement for a Survivor Claim

⁷A January 16, 1985 document from the West Virginia State Occupational Pneumoconiosis Fund records the payment of 104 weeks of benefits to Mrs. Bridges and indicates that her state widow's claim is "still in litigation" (DX 24-8). Eventually, Mrs. Bridges' West Virginia widow's claim was litigated before a state administrative law judge and resulted in a denial on November 30, 1994 (DX 6). Following the denial, Mrs. Bridges filed the Federal black lung survivor benefits claim (TR, page 2; see also employer's post-hearing brief, page 2).

Under the Act, and the implementing regulations, 20 C.F.R. §718.205, benefits are provided to eligible survivors of a miner whose death was due to pneumoconiosis. To obtain benefits, a surviving claimant must prove by a preponderance of the evidence several facts. First, the claimant must establish eligibility as a survivor. A surviving spouse may be considered eligible for benefits under the Act if he or she was married to, and living with, the coal miner at the time of his or her death and has not remarried.⁸

Next, the claimant must prove the coal miner had pneumoconiosis.⁹ In the regulation, “pneumoconiosis” is defined as a chronic dust disease arising out of coal mine employment. The definition further includes “any chronic pulmonary disease resulting in respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.”¹⁰ Under the Act, legal pneumoconiosis is much broader than medical pneumoconiosis. *Richardson v. Director, OWCP*, 94 F.3d 164 (4th Cir. 1996).

Third, once a determination has been made that a miner has pneumoconiosis, it must be determined whether the coal miner's pneumoconiosis arose, at least in part, out of coal mine employment.¹¹ If a miner who is suffering from pneumoconiosis was employed for ten years or more in one or more coal mines, there is a rebuttable presumption that pneumoconiosis arose out of such employment.¹² Otherwise, the claimant must provide competent evidence to establish the relationship between pneumoconiosis and coal mine employment.¹³

⁸20 C.F.R. §718.4 indicates the definitions in 20 C.F.R. §725.101 are applicable. 20 C.F.R. §725.101 in turn refers to the term “survivor” as used in Subpart B of Part 725. 20 C.F.R. §725.214 sets out the spousal relationship requirements and 20 C.F.R. §725.215 describes the dependency rules. According to §725.214 (a) the spousal relationship exists if the relationship is a valid marriage under state law. Under §725.215(a), a spouse is deemed dependent if he or she was residing with the miner at the time of his or her death.

⁹For survivor claims filed on or after January 1, 1982, an administrative law judge must make a threshold determination as to the existence of pneumoconiosis under 20 C.F.R. §718.202 (a) prior to determining whether a miner's death was due to pneumoconiosis under 20 C.F.R. §718.205. *Trumbo v. Reading Anthracite Company*, 17 B.L.R. 1-85 (1993).

¹⁰20 C.F.R. §718.201.

¹¹20 C.F.R. §718.203 (a).

¹²20 C.F.R. §718.203 (b).

¹³20 C.F.R. §718.203 (c).

Finally, the surviving spouse has to demonstrate the coal miner's death was due to pneumoconiosis. For a survivor claim filed on or after January 1, 1982, the Department of Labor regulations provide four means to establish that a coal miner's death was due to pneumoconiosis:¹⁴

1. Competent medical evidence establishes the death was caused by pneumoconiosis, or
2. Pneumoconiosis was a substantially contributing cause or factor leading to the miner's death, or
3. Death was caused by complications of pneumoconiosis, or
4. The presumption in 20 C.F.R. §718.304 regarding complicated pneumoconiosis applies.¹⁵

However, a survivor may not receive benefits if the coal miner's death was caused by traumatic injury or the principal cause of death was a medical condition not related to pneumoconiosis, unless evidence establishes that pneumoconiosis was a substantially contributing cause of death.¹⁶

Regarding the second method of establishing death due to pneumoconiosis, both the Benefits Review Board (“BRB”) and Federal courts of appeal have provided interpretations of the phrase, “substantially contributing cause or factor.”¹⁷ The BRB has stated a coal miner's death will be considered due to pneumoconiosis if the cause of the disease is significantly related to or significantly aggravated by pneumoconiosis. *Foreman v. Peabody Coal Company*, 8 B.L.R. 1-371, 1-374 (1985). The U.S. Court of Appeals for the Third Circuit has further broadened the interpretation by stating that any condition, such as pneumoconiosis, that hastens a coal miner's death is a “substantially contributing cause.” *Lukosevicz v. Director, OWCP*, 888 F.2d 1001, 1006 (3rd Cir. 1989). In reaching its decision, the court relied on the Department of Labor's comment when publishing Part 718 of the regulation that “pneumoconiosis need not be the 'principal,' sole, primary, or proximate cause of the miner's death in order for the survivor's claim to be compensable.” 48 Federal Register Page 24,277 at (n) (1983). In a similar case, the U.S. Court of Appeals for the Fourth Circuit adopted DOL's interpretation that pneumoconiosis substantially contributes to death if it hastens death in any way. *Shuff v. Cedar Coal Company*, 967 F.2d 977, 979 (4th Cir. 1992), *cert. denied*, 113 S. Ct. 969 (1993). In light of these judicial interpretations, if pneumoconiosis actually hastened a coal miner's death, then it is a substantially contributing cause within the meaning of the DOL regulations.

¹⁴20 C.F.R. §718.205 (c)(1), (2), and (3).

¹⁵Under this section, if there is evidence of complicated pneumoconiosis, then there is an irrebuttable presumption that the miner's death was due to pneumoconiosis.

¹⁶20 C.F.R. §718.205 (c)(4).

¹⁷20 C.F.R. §718.205 (c)(2).

In summary, a survivor's claim filed after January 1, 1982 must meet four, primary elements for entitlement. The claimant bears the burden of establishing these elements by a preponderance of the evidence. If the claimant fails to prove any one of the requisite elements, the claim for benefits must be denied. *Gee v. W. G. Moore and Sons*, 9 B.L.R. 1-4 (1986) and *Roberts v. Bethlehem Mines Corporation*, 8 B.L.R. 1-211 (1985). The four elements are: (1) the claimant is an eligible survivor of the deceased miner; (2) the coal miner suffered from pneumoconiosis; (3) the coal miner's pneumoconiosis arose out of coal mine employment; and, (4) the coal miner's death was due to coal workers' pneumoconiosis.

Eligible Survivor

In regards to the first element of entitlement, both the record (DX 7, DX 9, and DX 24-12) and Mrs. Bridges' uncontested testimony (TR, pages 14 and 15) establish that she married Mr. George Bridges in 1951, remained his wife through the date of his death, and has not since remarried. Consequently, I find Mrs. Mona T. Bridges is an eligible survivor under the Act.

Issue No. 1 - Presence of Pneumoconiosis

The second element that Mrs. Bridges must prove is that Mr. Bridges had pneumoconiosis. According to 20 C.F.R. §718.202, the existence of pneumoconiosis may be established by four methods: chest x-rays (§718.202 (a)(1)), autopsy or biopsy report (§718.202 (a)(2)), statutory presumption (§718.202 (a)(3)),¹⁸ or medical opinion (§718.202 (a)(4)). In addition, the U.S. Court of Appeals for the Fourth Circuit, based on its interpretation of the Act's language at 30 U.S.C. § 923 (b), requires that all the evidence found in each category be considered together to determine whether the preponderance of all relevant evidence supports a finding of pneumoconiosis. *Island Creek Coal Co. v. Compton*, 211 F.3d 203, 211 (4th Cir. 2000). In other words, rather than rendering a determination on each discrete subsection of 20 C.F.R. §718.202 (a), an administrative law judge must evaluate all the evidence before concluding whether a miner has pneumoconiosis.

The official record contains insufficient medical evidence to establish complicated pneumoconiosis and Mrs. Bridges filed her survivor claim well past the June 30, 1982 threshold date. As a result, the statutory presumptions of pneumoconiosis are not applicable. Accordingly, Mrs. Bridges must establish pneumoconiosis based on the totality of the chest x-ray evidence, autopsy or biopsy reports, and medical opinion.

¹⁸If any of the following presumptions are applicable, then under 20 C.F.R. §718.202 (a)(3) a miner is presumed to have suffered from pneumoconiosis: 20 C.F.R. §718.304 (if complicated pneumoconiosis is present, then there is an irrebuttable presumption that the miner's death was due to pneumoconiosis); 20 C.F.R. §718.305 (for claims filed before January 1, 1982, if the miner has fifteen years or more coal mine employment, there is a rebuttable presumption that total disability is due to pneumoconiosis); and 20 C.F.R. §718.306 (a rebuttable presumption when a survivor files a claim prior to June 30, 1982).

Chest X-Rays

The following table summarizes all the chest x-ray interpretations in the official record.

Date of X-Ray	Exhibit	Physician	Interpretation
January 1981 ¹⁹	DX 10	Leef, BCR ²⁰	Nodular fibrosis consistent with pneumoconiosis and overexpansion, most likely emphysema.
July 15, 1982	DX 10	(Reported by Dr. Mann)	Normal chest x-ray.
September 24, 1982	DX 11	Weinstein, BCR ²¹	Mild pulmonary emphysema. No infiltrates.
October 17, 1982	DX 11	Weinstein, BCR	Pulmonary emphysema. No infiltrates.
December 26, 1982	DX 11 and DX 24-21	Weinstein, BCR	Pulmonary emphysema, “the lungs are emphysematous, but free of infiltrate or vascular congestion.”
February 21, 1983	DX 11	Weinstein, BCR	Unchanged pulmonary emphysema. No infiltrates.

¹⁹The actual report is not in the record. Dr. Lapp summarized Dr. Leef’s findings (DX 10).

²⁰B - B Reader; and BCR - Board Certified Radiologist. These designations indicate qualifications a person may possess to interpret x-ray film. A “B Reader” has demonstrated proficiency in assessing and classifying chest x-ray evidence for pneumoconiosis by successful completion of an examination. A “Board Certified Radiologist” has been certified, after four years of study and an examination, as proficient in interpreting x-ray films of all kinds including images of the lungs.

As I informed the parties at the hearing, I take judicial notice of Dr. Leef’s board certification and have attached the certification documentation (TR, page 6).

²¹I take judicial notice of Dr. Weinstein’s board certification and have attached the certification documentation.

April 21, 1983	DX 11	Speiden, BCR ²²	Chronic obstructive pulmonary disease with suggested superimposed congestive changes.
November 2, 1983	DX 24-24	Gaziano, B	Positive for pneumoconiosis. Profusion category 1/0, ²³ type s opacities. ²⁴ Emphysema.
(same)	DX 24-25	Daniel, BCR ²⁵	Positive for pneumoconiosis. Profusion category 1/2, type u opacities.
March 31, 1984	DX 11	Weinstein, BCR	Pulmonary emphysema. Cardiovascular silhouette within normal limits.

Autopsy/Pathology

(Note: the following summary, and other remaining portions of this decision, contain detailed information obtained from the autopsy of Mr. Bridges, submitted by Mrs. Bridges to support her claim. While respecting the dignity and privacy of the deceased, some discussion of the detailed observations is necessary because I find the medical information relevant on determining whether Mr. Bridges' death was due to pneumoconiosis.)

Dr. Ahmed

²²I take judicial notice of Dr. Speiden's board certification and have attached the certification documentation.

²³The profusion (quantity) of the opacities (opaque spots) throughout the lungs is measured by four categories: 0 = small opacities are absent or so few they do not reach a category 1; 1 = small opacities definitely present but few in number; 2 = small opacities numerous but normal lung markings are still visible; and, 3 = small opacities very numerous and normal lung markings are usually partly or totally obscured. An interpretation of category 1, 2, or 3 means there are opacities in the lung which may be used as evidence of pneumoconiosis. If the interpretation is 0, then the assessment is not evidence of pneumoconiosis. A physician will usually list the interpretation with two digits. The first digit is the final assessment; the second digit represents the category that the doctor also seriously considered. For example, a reading of 1 / 2 means the doctor's final determination is category 1 opacities but he considered placing the interpretation in category 2. Or, a reading of 0/0 means the doctor found few or no opacities and didn't see a sufficient number of marks that would cause him or her to seriously consider category 1.

²⁴There are two general categories of small opacities defined by their shape: rounded and irregular. Within those categories the opacities are further defined by size. The round opacities are: type p (less than 1.5 millimeter (mm) in diameter), type q (1.5 to 3.0 mm), and type r (3.0 to 10.0 mm). The irregular opacities are: type s (less than 1.5 mm), type t (1.5 to 3.0 mm) and type u (3.0 to 10.0 mm). JOHN CRAFTON & ANDREW DOUGLAS, RESPIRATORY DISEASES 581 (3d ed. 1981).

²⁵I take judicial notice of Dr. Daniel's board certification and have attached the certification documentation.

Dr. M. Jamil Ahmed, board certified in anatomical and clinical pathology,²⁶ conducted an autopsy of Mr. Bridges on April 2, 1984 (DX 10 and 24-17).²⁷ Dr. Ahmed documented Mr. Bridges' coal mine employment and 20 year history as a cigarette smoker. Gross examination of the chest area disclosed left ventricular myocardial hypertrophy and the thoracic aorta had "many plaques of arteriosclerosis." Dr. Ahmed reported the lungs displayed "an impressive degree of emphysema and a stage of simple coal workers' pneumoconiosis." The emphysema was present throughout the lungs. Blackish pneumoconiosis macules, measuring up to 3 millimeters, were present, but not in any conglomerations.

Dr. Ahmed's microscopic examination of the arteries indicated coronary arteriosclerosis and left ventricular myocardial hypertrophy. The coal macules in the lung tissue were consistent with simple coal workers' pneumoconiosis and contained "dusty deposits of coal and/or anthracotic pigment." The macules were haphazardly distributed throughout the lung tissue. The tracheobronchial tissue also contained coal macules, some with extensive fibrosis. Finally, Dr. Ahmed also observed pulmonary congestion and edema.

Based on the autopsy findings, Dr. Ahmed diagnosed simple coal worker's pneumoconiosis, "pneumoconiosis with tracheobronchial lymph node involvement," emphysema, and pulmonary edema. Dr. Ahmed also observed evidence of a left ventricular myocardial "hypertrophy" and "coronary arteriosclerosis."

Dr. Rodman

About the beginning of 1990,²⁸ Dr. Nathaniel F. Rodman, then chairman of pathology at West Virginia University, reported his microscopic findings of Mr. Bridges' autopsy slides (DX 10). He found 40% of the lung tissue to be normal and another 40% involved with emphysema. In the sections containing fibrotic emphysema, Dr. Rodman also observed the deposition of small to moderate amounts of black pigment in 30% of the material. Concerning the dust particle deposit, Dr. Rodman opined that deposits occurred due to the emphysema fibrosis. Specifically, the pigment was "a secondary deposit rather than the pigment being a primary deposit with secondary damage to the pulmonary" tissue.

In the remaining 20% of the lung tissue, Dr. Rodman saw a few macules and micro-nodules, measuring up to 7 millimeters in diameter, consistent with coal workers' pneumoconiosis, or

²⁶I take judicial notice of Dr. Ahmed's board certification and have attached the certification documentation.

²⁷Other than the cover sheet containing Dr. Ahmed's preliminary anatomic diagnosis, the original report is missing. However, Dr. Rodman, in his review of the report, summarized its contents (DX 10). In addition, due to the lapse of time and hospital protocol, the pathology slides relating to Mr. Bridges' autopsy have been destroyed (EX 4).

²⁸Although Dr. Rodman's report is undated, he refers to a December 26, 1989 letter from an attorney who provided copies of Mr. Bridges' medical record.

anthracosilicosis. He described these areas as “dense collagenous connective tissue. . . containing dense deposits of coal mine dust.”

Under the microscope, the bronchial lymph nodes had “several small foci of collagenous connective tissue. . . with very tiny silicotic nodules as well as mild to moderate deposition of black pigment. . .” These deposits were not pulmonary in nature. Instead, Dr. Rodman characterized the nodules as “non-pulmonary lesions” representing “drainage of particles from the lung into a depository of which the lymph nodes constitute a major reservoir.”

The microscopic sections of the heart tissue did not reveal any evidence of an infarction or hypertrophy. However, the coronary artery sections showed moderate atherosclerosis.

Medical Opinion

Dr. Previll and Dr. Leef

In January 1981, Dr. J. M. Previll, board certified in internal medicine,²⁹ and Dr. J. L. Leef, Jr., board certified in diagnostic radiology, conducted a pulmonary examination of Mr. Bridges (DX 10). The physicians noted his 36 years of coal mine employment and a cigarette smoking history of 20 pack years.³⁰ Mr. Bridges continued to work in the mines but was taking medication to assist his breathing. On physical examination, the doctors observed Mr. Bridges used neck muscles to assist his deep breathing. The breath sounds were decreased, with bilateral wheezing. After exercise, Mr. Bridges had moderately labored breathing and hyperventilation. The pulmonary function test showed the presence of a moderate obstructive disease. Dr. Previll and Dr. Leef diagnosed occupational pneumoconiosis with no change in the 25% disability rating established in 1975.

Dr. Mann

Between September 24, 1982 and March 22, 1984, Mr. Bridges was treated for shortness of breath by Dr. Thomas F. Mann, board certified in internal medicine³¹ (DX 10).³² In 1982, the shortness of breath was persistent but mild according to objective medical testing. Dr. Mann believed the breathing problem might have a psychosomatic component. However, Dr. Mann placed Mr. Bridges on Prednisone and oxygen therapy. Although Mr. Bridges responded well to the treatments, he still experienced numerous

²⁹I take judicial notice of Dr. Previll’s board certification and have attached the certification documentation.

³⁰A pack year represents the consumption of a pack of cigarettes a day for one year.

³¹I take judicial notice of Dr. Mann’s board certification and have attached the certification documentation.

³²In 1982, Dr. Joseph Shaver also treated Mr. Bridges at the same clinic on three occasions. His observations were consistent with Dr. Mann’s reports.

bouts of shortness of breath. A year later, by December 1983, the emphysema seemed to be progressing and Mr. Bridges' pulmonary function tests were poor.

During approximately the same period, Mr. Bridges was hospitalized on several occasions for pulmonary emphysema and chronic asthma.³³ He typically received intravenous bronchodilators, steroids, antibiotics, and occasional oxygen assistance during these hospitalizations. For example, on December 26, 1982, Mr. Bridges arrived at the emergency room of the Greenbrier Valley Hospital with extreme shortness of breath (DX 10 and DX 24-21). Use of a nebulizer provided temporary relief, but Mr. Bridges was admitted to the hospital for observation. During the hospital stay, Dr. Mann examined Mr. Bridges and heard poor breath sounds. A blood gas study revealed values that did not qualify as totally disabling under the black lung regulations.³⁴ A chest x-ray indicated pulmonary emphysema. Dr. Mann noted that Mr. Bridges had been previously discharged from the hospital on October 25, 1982 after treatment for asthma and most of his medical history concerned his asthma. He also indicated that Mr. Bridges had worked as a coal miner and quit smoking cigarettes about 1977. Based on his examination, Dr. Mann diagnosed chronic asthma "made worse by . . . pulmonary emphysema." Eventually, in January 1983, Dr. Mann submitted to DOL a copy of his December 1982 evaluation and some additional blood gas studies from October 1982 which indicated an oxygen exchange impairment and pulmonary function test revealing a severe obstructive defect that improved with application of a bronchodilator.

On February 25, 1994, Dr. Mann prepared a statement emphasizing Mr. Bridges' condition in relation to occupational pneumoconiosis (DX 6).³⁵ Dr. Mann summarized Mr. Bridges' demise as follows:³⁶

Mr. Bridges had significant pulmonary problems which were the result partly of intrinsic asthma and in part by coal workers pneumoconiosis. The pneumoconiosis aspect of this made his asthma more difficult to treat and he had numerous admissions over the last couple of years of his life. He died of an unexpected cardiac arrest while hospitalized for respiratory failure. While his immediate cause of death was most certainly some type of heart arrhythmia, this was certainly induced by his respiratory failure and the primary cause for his respiratory failure was his pneumoconiosis.

³³Specifically, July 15 to 19, 1982; September 24 to 28, 1982; October 17 to 25, 1982; December 26 to 31, 1982; February 21 to 25, 1983; and April 21 to 26, 1983 (DX 10).

³⁴To qualify for Federal Black Lung disability benefits, at a coal miner's given $p\text{CO}_2$ level, the value of the coal miner's $p\text{O}_2$ must be equal to or less than corresponding $p\text{O}_2$ value listed in the Blood Gas Tables in Appendix C for 20 C.F.R. §718.

³⁵The actual document is not in the record. Dr. Walker, in his testimony before a West Virginia administrative law judge, referenced the contents of the report (DX 6).

³⁶Again, since Dr. Mann's 1994 report is not in the record, the source of this direct quote is the state compensation appeal brief submitted by claimant's counsel at the time, Mr. William Turner (DX 10).

Dr. Daniel

On November 2, 1983, in Beckley, West Virginia, Dr. John M. Daniel, board certified in family practice,³⁷ conducted a pulmonary examination of Mr. Bridges (DX 24-22). Without mentioning Mr. Bridges' coal mine employment history, Dr. Daniel noted a cigarette smoking history of about 15 years at up to one pack of cigarettes a day. Mr. Bridges also reported a "touch of asthma" and numerous hospitalizations for shortness of breath since July 1982. During the physical examination, Dr. Daniels heard wheezing and rales. A pulmonary function test, found acceptable by Dr. Gaziano (DX 24-20), produced the following results: FEV₁ - 0.89; FVC - 2.61; MVV - 30; and FEV₁/FVC of 34%. Dr. Daniel believed the test showed a moderate restrictive defect and severe obstructive defect. Blood gas studies revealed a mild degree of hypoxemia which improved with exercise. A chest x-ray was positive for pneumoconiosis. Dr. Daniel's diagnosis included pneumoconiosis related to coal dust exposure and chronic obstructive pulmonary disease ("COPD") that was not due to coal dust. Finally, Dr. Daniel opined Mr. Bridges could still perform his usual activities as a coal miner.

Dr. Modlin

On March 31, 1984, Mr. Bridges arrived at the hospital in Greenbrier, West Virginia with wheezing and subjective signs of shortness of breath (DX 10 and DX 11). Dr. Robert K. Modlin, board certified in internal medicine,³⁸ examined Mr. Bridges. Other than wheezing, the physical examination was normal. The chest x-ray indicated emphysema. Dr. Modlin hospitalized Mr. Bridges, noting he was being treated by Dr. Mann for shortness of breath. Dr. Modlin observed that Mr. Bridges seemed to have his greatest breathing difficulties in the spring. Dr. Modlin continued Mr. Bridges on his breathing medication and provided oxygen therapy. During that evening, Mr. Bridges was restless and unable to sleep. At 5:00 a.m. on April 1, 1984, Mr. Bridges suffered cardiac arrest and could not be revived.

On April 1, 1984, Dr. Modlin provided a final diagnosis of "chronic obstructive pulmonary disease with respiratory failure (black lung)" and arteriosclerotic heart disease with "presumed myocardial infarction." On another hospital record, Dr. Modlin checked a box indicating no autopsy had been accomplished (DX 11).³⁹ He also signed Mr. Bridges death certificate, indicating Mr. Bridges died on April 1, 1984 due to "black lung (COPD)" while an inpatient at the Greenbrier Valley Hospital (DX 8, 9, and DX 24-15). He also listed myocardial infarction as a significant condition contributing to death and noted no autopsy had been performed.

³⁷I take judicial notice of Dr. Daniel's board certification and have attached the certification documentation.

³⁸I take judicial notice of Dr. Modlin's board certification and have attached the certification documentation.

³⁹Although Dr. Modlin dictated the hospital discharge summary on April 9, 1984, there is no indication he reviewed Dr. Ahmed's autopsy report.

Dr. Lapp

On March 17, 1987, Dr. N. Leroy Lapp, board certified in pulmonary disease and internal medicine,⁴⁰ conducted a review of the medical evidence in Mr. Bridges' case (DX 10).⁴¹ Dr. Lapp believed the 1983 pulmonary function studies showed a severe air flow obstruction. He also referred to Dr. Preville's and Dr. Leef's notation of Mr. Bridges' cigarette smoking history and use of neck muscles to assist his breathing. Dr. Leef found the chest x-ray positive for pneumoconiosis and most likely emphysema. Clearly, by x-ray, Mr. Bridges had occupational pneumoconiosis, and clinical examinations, and pulmonary function studies established the presence of "diffuse pulmonary emphysema." Likewise, the autopsy report established the presence of diffuse emphysema and pneumoconiosis. Based on this medical information and medical studies concerning pneumoconiosis induced mortality, Dr. Lapp opined "it is unlikely that pneumoconiosis was the cause of Mr. Bridges' death." He believed the coronary artery disease and emphysema were more important factors than pneumoconiosis in causing Mr. Bridges to die. Due to the left ventricle hypertrophy, he also surmised that Mr. Bridges died of a myocardial infarction.

Dr. Rodman

In addition to his pathology report, Dr. Rodman also reviewed Mr. Bridges' medical record, including Dr. Ahmed's autopsy report (DX 10). Based on this objective medical evidence, he characterized Mr. Bridges' cause of death as "complex." According to Dr. Rodman:

the actual mechanism of Mr. Bridges' death. . . was almost certainly a cardiac arrhythmia going through ventricular tachycardia to ventricular fibrillation and finally death. There is no evidence to suggest that coal workers' pneumoconiosis can produce such an arrhythmia leading to death. Again, it is the opinion of this reviewer that the most important causes in the death of Mr. Bridges were his bronchial asthma or asthmatic bronchitis and his severe panacinar emphysema. It is my opinion that Mr. Bridges would have died at the same time he did and by the same mechanism he did whether or not he had coal workers' pneumoconiosis. There is no evidence that either the asthmatic bronchitis o[r] the

⁴⁰I take judicial notice of Dr. Lapp's board certification and have attached the certification documentation.

⁴¹Dr. Lapp's actual report is not in the record. However, Dr. Rodman, during his medical record review (DX 10) summarized its contents.

panacinar emphysema had any cause and effect relationship with Mr. Bridges' coal mining occupation.

Dr. Rodman explained that although Mr. Bridges responded to treatments for asthma, he continued to require periodic hospitalization due to shortness of breath associated with his asthma. In addition, Mr. Bridges suffered severe and extensive emphysema. Even though he had stopped smoking several years earlier, the emphysema was caused by Mr. Bridges' "prolonged and protracted cigarette smoking." While coal workers' pneumoconiosis "may have been a contributing factor," it was minor in comparison with the significant asthma and emphysema. Concerning the heart, the ventricular hypertrophy was caused by the emphysema. And, while Mr. Bridges also had coronary artery arteriosclerosis, it was not severe enough to be clinically significant or a cause of death.

Dr. Walker

At a March 2, 1994 hearing before a state of West Virginia administrative law judge,⁴² Dr. James H. Walker, a board certified surgeon⁴³ and chairman of the Occupational Pneumoconiosis Board ("Board") that initially determined on March 14, 1985 pneumoconiosis was a major contributing factor in Mr. Bridges' death, explained the objective medical evidence that caused him to change his opinion and conclude that pneumoconiosis did not play any role in Mr. Bridges' death (DX 6). At the time of the 1994 hearing, Dr. Walker opined pneumoconiosis neither caused, nor contributed to, Mr. Bridges' death.

First, Dr. Walker reviewed Dr. Mann's earlier reports and diagnoses of Mr. Bridges' severe asthma and heart disease. Mr. Bridges suffered numerous asthma attacks with subsequent periods of improvement. Although Dr. Mann also provided a statement in February 1994 that supported a finding that pneumoconiosis was a contributing factor, Dr. Walker did not rely on that assessment because Dr. Mann apparently did not address Mr. Bridges' asthma. Second, Dr. Rodman's review of the autopsy pathology indicated "findings typical of asthma, congenital heart disease, and little pneumoconiosis." According to Dr. Walker, the pathology report disclosed Mr. Bridges' lung tissue contained "a few macules and micronodules measuring up to 7 millimeters in their greatest diameter." Third, based on the pathology report, Dr. Walker no longer considered Mr. Bridges' pneumoconiosis significant. Fourth, when the Board rendered its 1985 finding of sufficient pneumoconiosis, the members were not aware of the amount of Mr. Bridges' bronchial asthma and the "very insignificant" amount of pneumoconiosis found during the autopsy. Fifth, Dr. Walker found no causal connection between the pneumoconiosis and the asthma.

Dr. Hayes

⁴²Ms. Bridges was represented by counsel at the hearing (DX 6).

⁴³I take judicial notice of Dr. Walker's board certification and have attached the certification documentation.

In the March 1994 state administrative law judge hearing, Dr. Thomas Hayes, board certified in radiology⁴⁴ and a current member of the Board, also addressed the Board's revised assessment concerning Mr. Bridges' cause of death (DX 6). Dr. Hayes commented that Mr. Bridges was relatively symptom free between his bouts with asthma. In addition, his shortness of breath typically occurred in the spring, which is consistent with asthma. Mr. Bridges' history of asthma was not presented to the March 1985 Board prior to its initial determination. Dr. Hayes stated the Board now believed occupational pneumoconiosis did not cause or contribute to a material degree to Mr. Bridges' death.

Dr. Pushkin

At the March 1994 state administrative law judge hearing, Dr. Willard Pushkin, a member of the March 1985 Board that found pneumoconiosis contributed to Mr. Bridges' death, concurred with Dr. Walker's and Dr. Hayes' revised medical opinions that occupational pneumoconiosis did not materially cause, or contribute to, Mr. Bridges' death (DX 6).

Dr. Gaziano

In October 1998, Dr. D. Gaziano, board certified in pulmonary disease and internal medicine,⁴⁵ responded to an inquiry by DOL on whether the pneumoconiosis established by the autopsy report was either the cause of, or a contributing factor to, Mr. Bridges' death. Dr. Gaziano responded that Mr. Bridges suffered a respiratory death with pneumoconiosis being a significant contributing factor. He based his conclusion in part on the autopsy/pathology finding of pneumoconiosis in 20% of the lung tissue. In addition, Mr. Bridges entered the hospital in respiratory distress with a history of chronic obstructive pulmonary disease and asthmatic bronchitis. His pulmonary function tests showed moderately severe pulmonary impairment. As a result, Mr. Bridges died due to prolonged respiratory distress and a subsequent cardiac arrest.

In a July 9, 1999 deposition (EX 6), Dr. Gaziano reiterated his assessment that Mr. Bridges suffered a respiratory death. Even though the blood gas study obtained upon his hospital admission showed only a mild hypoxemia, Mr. Bridges experienced a prolonged respiratory difficulty which represents a strain on his heart that can cause arrhythmia. In addition, Mr. Bridges did have a heart disease.

Dr. Gaziano believed the autopsy finding of 20% pneumoconiosis was consistent with his 1983 x-ray finding of pneumoconiosis. The degree of pneumoconiosis was mild to moderate. Dr. Gaziano acknowledged that he attributed Mr. Bridges' death in part to pneumoconiosis because Mr. Bridges had coal workers' pneumoconiosis and a significant pulmonary impairment.

⁴⁴I take judicial notice of Dr. Hayes' board certification and have attached the certification documentation.

⁴⁵I take judicial notice of Dr. Gaziano's board certification and have attached the certification documentation.

Dr. Gaziano did not think coal dust contributed to Mr. Bridges' asthma. Mr. Bridges also had a chronic obstructive pulmonary disease ("COPD") and bronchitis. In Mr. Bridges' case, where there are two possible causes for impairment, coal workers' pneumoconiosis and COPD/bronchitis, Dr. Gaziano is not able to exclude either cause as a contributing factor to a respiratory impairment. At the same time, Dr. Gaziano acknowledged that he has treated individuals for severe COPD who only had an extensive history of cigarette smoking and never mined coal.

While coal dust may cause emphysema, it typically produces focal emphysema. On the other hand, cigarette smoke will produce diffuse central lobular emphysema. On the subject of Mr. Bridges' emphysema, Dr. Gaziano was asked, "Do you think that it [coal mine dust exposure] is capable of causing the kind of panacinar emphysema that was mentioned by Dr. Rodman in his report?" Dr. Gaziano stated, "I don't think so, I don't think it produces the severity of emphysema we see in this case."

Dr. Koenig

On July 19, 1999, Dr. Steven M. Koenig, board certified in pulmonary disease and internal medicine, provided a medical opinion based on a review of Mr. Bridges' x-rays and medical record, Dr. Ahmed's autopsy report, and the medical reviews of Dr. Rodman, Dr. Kleinerman, Dr. Caffrey, and Dr. Hutchins (CX 1). Dr. Koenig also noted Mr. Bridges' coal mine employment of nearly 38 years. Dr. Koenig found the medical evidence established Mr. Bridges had extensive and severe panacinar emphysema, which caused his severe airflow obstruction and is labeled chronic obstructive pulmonary disease ("COPD"). COPD includes both emphysema and chronic bronchitis. At the same time, even though the COPD was reversible with bronchodilators, Dr. Koenig found such reversal consistent with COPD and he agreed with Dr. Kleinerman that Mr. Bridges did not have asthma. Consequently, Mr. Bridges had severe COPD which was totally disabling. Contrary to the opinions of other physicians, Mr. Bridges' pulmonary condition was permanent. Since the emphysema was permanent and not reversible, he did not have a normal respiratory capacity between his hospitalizations.

The autopsy and pathology reports demonstrate that Mr. Bridges had "classic" simple coal workers' pneumoconiosis. This type of pneumoconiosis only causes a restrictive impairment. According to the medical tests, Mr. Bridges did not have any restrictive pulmonary impairment. Consequently, the pneumoconiosis found in Mr. Bridges' lungs after his death did not cause any "clinically significant pulmonary impairment." Also, the classic pneumoconiosis "did not contribute significantly to Mr. Bridges' severe pulmonary impairment."

Clearly cigarette smoking is responsible for Mr. Bridges' COPD and corresponding severe pulmonary impairment. At the same time, "exposure to coal dust was a possible cause and/or a contributing factor to Mr. Bridges' COPD as well." To support his position, Dr. Koenig refers to medical studies which indicate COPD can develop as a result of coal dust exposure independent of a miner's cigarette smoking history. Such coal dust induced COPD can be severe and disabling. In addition, the

presence of classic coal workers' pneumoconiosis in Mr. Bridges' lungs increases the likelihood that coal dust also contributed to the COPD.

The immediate cause of Mr. Bridges' death was cardiac arrhythmia. However, the narrowing of Mr. Bridges' arteries was not great enough to cause heart failure. Rather, the severe COPD, by causing hypoxemia, can induce heart failure. Even though Mr. Bridges' blood gas study upon admission on March 31, 1984 did not show severe hypoxemia, Mr. Bridges later experienced restlessness and insomnia, clinical evidence of hypoxemia and respiratory failure. And even if the COPD was not the primary cause of the heart failure, "[a]t the very least, Mr. Bridges' COPD contributed to his death." Any assertion that COPD did not play a role in Mr. Bridges' death, considering the circumstances of his death is "ludicrous." In summary, Dr. Koenig opined that "coal mine employment caused or at least contributed to his severe respiratory symptoms, impairment, total disability and death."

Dr. Kleinerman

On March 13, 1999, Dr. Jerome Kleinerman, a board-certified anatomic and clinical pathologist, conducted a review of all the medical evidence in Mr. Bridges' case including the autopsy and pathology reports and hospitalization records (EX 1).⁴⁶ Dr. Kleinerman also considered Mr. Bridges' coal mine employment and cigarette smoking histories.

After his review, Dr. Kleinerman first concluded that Mr. Bridges had simple coal workers' pneumoconiosis and there was no evidence of complicated pneumoconiosis. Mr. Bridges' breathing problems were not caused by simple coal workers' pneumoconiosis. This pneumoconiosis did not have any role in Mr. Bridges' death.

Mr. Bridges suffered periodic bouts of severe bronchospasm caused by asthmatic bronchitis and severe emphysema, which are unrelated to the simple coal workers' pneumoconiosis. Yet, despite these pulmonary problems, early medical tests show near normal pulmonary functions. And, between his periodic bouts of bronchospasms, Mr. Bridges probably had near normal respiration.

Next, in regards to Dr. Rodman's observation of the emphysema tissue, Dr. Kleinerman characterizes Dr. Rodman's findings as a "nonspecific interstitial fibrosis with airspace enlargement." This type of emphysema was caused by his use of cigarettes. In fact, medical studies confirm that cigarette smoking for 20 years, a pack a day, or more, is the most common and important cause of panacinar emphysema and chronic bronchitis.

Concerning Mr. Bridges' death, Dr. Kleinerman, while agreeing the immediate cause of death was cardiac arrhythmia, is unable to ascertain the actual cause. He eliminates respiratory failure as a cause, because the results of Mr. Bridges' blood gas study at the time of admission were not consistent with

⁴⁶Dr. Kleinerman confirmed on July 19, 1999 that Dr. Gaziano's July 9, 1999 deposition did not alter his opinion (EX 6).

respiratory failure. And, the autopsy eliminated congestive heart failure and clogged coronary arteries as possible causes.

Dr. Kleinerman concludes that neither simple pneumoconiosis nor exposure to coal dust caused, contributed, or hastened Mr. Bridges' death.

Dr. Caffrey

On April 19, 1999, Dr. P. Raphael Caffrey, board certified in anatomical and clinical pathology, presented a medical opinion concerning the death of Mr. Bridges (EX 2). After considering Mr. Bridges' work, cigarette smoking, and medical histories, the hospitalization records, associated medical tests, autopsy and pathology reports, and consultation reports, Dr. Caffrey concluded that Mr. Bridges had simple pneumoconiosis caused by his coal mine employment. Since coal workers' pneumoconiosis was present in only 20 % of the lungs and didn't show up on the most recent x-rays, it was not extensive or severe. Pneumoconiosis did not cause, play any significant role in, or hasten Mr. Bridges' death.

Mr. Bridges also had extensive panacinar emphysema due to a significant smoking history, and asthmatic bronchitis. The later two pulmonary conditions were not caused by coal mine employment.

Dr. Caffrey observed that Dr. Ahmed found evidence of coronary artery arteriosclerosis and left ventricular myocardial hypertrophy. These heart problems, while not caused by coal mine employment, could have contributed to Mr. Bridges' heart arrhythmia.

In a September 17, 1999 deposition, Dr. Caffrey explained the three stages of coal workers' pneumoconiosis (EX 10). The first stage consists of a lesion containing anthracotic pigment or coal dust with a tissue reaction of reticulin which forms a focal emphysema. In the second stage, micro- and macro-nodules develop. And the third stage is complicated pneumoconiosis. Dr. Caffrey believes Mr. Bridges has a "mild" case of coal workers' pneumoconiosis.

There are two general types of emphysema: centriacinar and panacinar. Focal emphysema is a form of centriacinar emphysema which is located in the center of lobule. Panacinar emphysema is "peripheral-oriented" and usually occurs in the lower lobes. Mr. Bridges had extensive and severe panacinar emphysema.

Mr. Bridges also had heart disease in the form of coronary artery atherosclerosis and enlargement of the left side of the heart, which is typically associated with coronary atherosclerosis. Based on the hospitalization records from March 31, 1984 and Dr. Rodman's findings, Dr. Caffrey believes Mr. Bridges suffered a cardiac death. The amount of simple coal workers' pneumoconiosis in Mr. Bridges' lungs would not have caused the cardiac arrhythmia.

Dr. Caffrey disagrees with Dr. Gaziano's assessment that pneumoconiosis contributed to death simply because Mr. Bridges had pneumoconiosis and a pulmonary impairment. Likewise, he believes Dr. Koenig's assertion that the coal dust contributed to Mr. Bridges' death is incorrect because Dr. Koenig relied, in part, on the emphysema as a contributing factor. Although Mr. Bridges did have emphysema, that type of pulmonary disease, panacinar emphysema, is caused by cigarette smoke and not coal dust. Coal dust causes focal or centriacinar emphysema. Consequently, Dr. Caffrey reaffirms his conclusion that coal dust exposure did not hasten death in this case.

On cross-examination, Dr. Caffrey stated that if someone, who had both smoked cigarettes and worked in a coal mine, developed COPD, he would not be able to distinguish the source of the COPD.

Dr. Hutchins

In 1999, Dr. Grover M. Hutchins, board certified in anatomic and pediatric pathology, also conducted a medical review of Mr. Bridges' case in a manner similar to Dr. Caffrey (EX 3, EX 6, and EX 8). According to Dr. Hutchins, Mr. Bridges had a mild case of coal workers' pneumoconiosis since most of the x-rays didn't disclose its presence and the pathology study only found it in 20% of the tissue. His coal workers' pneumoconiosis was insufficient to cause, or contribute to, a respiratory impairment. Instead, Mr. Bridges suffered a pulmonary impairment due to panacinar emphysema and asthmatic bronchitis which produced COPD. The COPD was "induced by cigarette smoke and superimposed asthma. Neither coal dust exposure or coal workers' pneumoconiosis hasten or contribute to Mr. Bridges' death.

Dr. Castle

Dr. James R. Castle, board certified in pulmonary disease and internal medicine, also conducted a medical review of Mr. Bridges' case in the summer of 1999 (EX 5 and EX 6). Based on the medical evidence and pathology reports, Dr. Castle concluded Mr. Bridges did have simple coal workers' pneumoconiosis. Because the x-ray failed to confirm its presence, as established by pathology examination, the black lung disease was "mild."

Mr. Bridges also struggled with asthmatic bronchitis. However, there is no relationship between his asthma and coal mining. In addition, according to the autopsy and pathology reports, Mr. Bridges had panacinar emphysema caused by Mr. Bridges' "long and extensive history of tobacco abuse."

Because Mr. Bridges' blood gas studies at the time of admission were near normal, there is no medical evidence of impending respiratory failure. On the other hand, there is evidence of coronary artery disease. Individuals with coronary artery disease and asthmatic bronchitis may experience cardiac arrhythmia. In light of the autopsy findings, Dr. Castle opines Mr. Bridges died as the result of "untreated pulmonary edema that resulted in cardiac arrhythmia."

The autopsy and pathology report “clearly documents that he has tobacco-induced pulmonary emphysema of a severe degree as well as minimal changes of coal workers’ pneumoconiosis.”

In a July 28, 1999 deposition, Dr. Castle presented several points (EX 7). First, coal workers’ pneumoconiosis played no role in Mr. Bridges’ death. Second, he disagrees with Dr. Koenig’s opinion that coal dust related COPD contributed to Mr. Bridges’ death and disputes his interpretation of his cited medical studies. Third, to identify COPD exacerbation, mentioned by Dr. Koenig, physicians must rely on objective medical evidence, since subjective complaints of shortness of breath are insufficient to identify the cause of the breathing problem. In Mr. Bridges’ situation, the blood gas studies and physical examination were near normal. Consequently, Dr. Castle does not believe a diagnosis of severe COPD exacerbation is credible. Fourth, the autopsy disclosed signs of left ventricular hypertrophy and pulmonary edema, which is caused by coronary artery disease. As a result, Dr. Castle concludes the cardiac arrhythmia was due to Mr. Bridges’ cardiac disease. Fifth, Dr. Castle would agree that cigarette smoke was not the sole cause of Mr. Bridges’ COPD and coal dust did contribute to the COPD.

Discussion

The regulatory definition of pneumoconiosis includes “any chronic pulmonary disease resulting in respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.”⁴⁷ Due to this definition, lung disease, not ordinarily considered pneumoconiosis in medical terms, may be pneumoconiosis in legal terms if coal caused or aggravated the condition. See *Richardson v. Director, OWCP*, 94 F.3d 164 (4th Cir. 1996). In other words, regulatory pneumoconiosis includes both “medical” and “legal” pneumoconiosis. Consequently, my discussion on the presence of pneumoconiosis must cover both medical pneumoconiosis and legal pneumoconiosis.

Medical Pneumoconiosis

As explained by Dr. Castle, the second stage of typical pneumoconiosis involves the development of micro- and macro-nodules around coal dust deposits as the lung tissue responds to the deposit of coal dust. These nodules may then appear on chest x-rays. I believe this process defines medical pneumoconiosis, also referred to as “classic” or “simple” pneumoconiosis.

Turning first to the radiographic evidence, of the nine chest x-rays, only two films were interpreted as positive for pneumoconiosis. The other seven x-rays, including the x-ray taken the night before Mr. Bridge died, did not yield a positive finding of pneumoconiosis. Standing alone, the preponderance of the chest x-ray evidence does not establish the presence of pneumoconiosis.

⁴⁷20 C.F.R. §718.201.

On the other hand, the autopsy and pathology reports by Dr. Ahmed and Dr. Rodman document the presence of classic coal workers' pneumoconiosis in Mr. Bridges' lung tissue. Almost all the physicians who reviewed this case agreed with the two pathologists' findings that Mr. Bridges had black lung disease. These same physicians opined that the radiographic evidence, rather than being inconsistent, helped determine the extent of the pneumoconiosis. While the pathologists found black lung disease scattered throughout about 20% of the lung tissue, the pneumoconiosis was not sufficiently extensive to appear on radiographic films. As a result, the physicians concluded Mr. Bridges had a "mild" case of black lung disease. In other words, absence of x-ray evidence of the disease was reasonable and not inconsistent with the pneumoconiosis found in the lung tissue samples. In light of the consensus of medical opinion on the presence of pneumoconiosis, the explanation regarding the absence of positive chest x-ray interpretations, and considering that a direct microscopic examination of lung tissue may be more probative than a chest x-ray,⁴⁸ I find the preponderance of the more probative evidence establishes that Mr. Bridges had a mild case of "classic" black lung disease in his lungs. Based on my finding, Mrs. Bridges is able to prove the second requisite element for a survivor claim, the presence of pneumoconiosis in her husband's lungs.

Legal pneumoconiosis

In addition to the classic black lung disease, medical evidence suggests Mr. Bridges may have struggled with two other pulmonary disorders. Dr. Ahmed and Dr. Rodman found evidence of emphysema during their pathology examinations. And, Dr. Mann and several other physicians diagnosed asthma or asthmatic bronchitis.

As mentioned above, the legal definition of pneumoconiosis extends to any lung impairment that is related to, or aggravated by, coal dust exposure. Consequently, I must determine whether Mr. Bridges' asthma or emphysema has any connection with his coal mine employment.

Asthma or Asthmatic Bronchitis

Of all the physicians in this case who addressed the cause of Mr. Bridges' asthma/asthmatic bronchitis, Dr. Mann stands alone in finding a connection between Mr. Bridges' asthma and his coal mine employment. According to Dr. Mann, Mr. Bridges' exposure to coal dust made his asthma more difficult to treat. Because that statement implies coal dust may have aggravated Mr. Bridges' respiratory condition of asthma, Dr. Mann's opinion supports a finding of legal pneumoconiosis.

On the other hand, numerous physicians disagreed with Dr. Mann's conclusion. Dr. Rodman found insufficient pathology evidence to link Mr. Bridges' asthmatic bronchitis to his coal mine employment.

⁴⁸See *Terlip v. Director, OWCP*, 8 B.L.R. 1-363 (1985). Autopsy evidence is the most reliable evidence of the existence of pneumoconiosis and carries significant probative weight.

Likewise, Dr. Gaziano, Dr. Kleinerman, Dr. Caffrey and Dr. Castle concurred with Dr. Rodman's assessment.

Because there is a conflict of opinion among the medical experts on whether Mr. Bridges' asthma/asthmatic bronchitis was connected to his coal dust exposure and amounted to regulatory pneumoconiosis, I must initially assign relative probative weight to the diverse conclusions. In evaluating medical opinions, an administrative law judge must first determine whether opinions are based on objective documentation and then consider whether the conclusions are reasonable in light of that documentation. A well-documented opinion is based on clinical findings, physical examinations, symptoms, and a patient's work history. See *Fields v. Island Creek Coal Company*, 10 B.L.R. 1-19 (1987) and *Hoffman v. B&G Construction Company*, 8 B.L.R. 1-65 (1985). For a medical opinion to be "reasoned," the underlying documentation and data should be sufficient to support the doctor's conclusion. See *Fields*, 10 B.L.R. 1-19 (1987). In evaluating conflicting medical reports, it may be appropriate to give more probative weight to the most recent report. See *Clark v. Karst Robbins Coal Company*, 12 B.L.R. 1-149 (1989)(en banc). At the same time, "recency" by itself may be an arbitrary benchmark. See *Thorn v. Itmann Coal Company*, 3 F.3d 713 (4th Circuit 1993). But, the qualifications of the doctor who provided the most recent evaluation may also bear on the evidentiary weight of the study. See *McMath v. Director, OWCP*, 12 B.L.R. 1-6 (1988). Finally, a medical opinion may be given little weight if it is vague or equivocal. See *Griffith v. Director, OWCP*, 49 F.3d 184 (6th Circuit 1995) and *Justice v. Island Creek Coal Company*, 11 B.L.R. 1-91 (1988).

With these principals in mind, I recognize that Dr. Mann, as Mr. Bridges' treating physician, had a unique opportunity to develop a well documented and reasoned medical opinion. With greater probative weight, his sole opinion that the asthma was related to coal dust may have outweighed the consensus opinion of the other doctors. However, in this case, I do not give Dr. Mann's opinion increased probative weight for two principal reasons. First, the record does not indicate whether he reviewed Dr. Ahmed's and Dr. Rodman's pathology findings, which the other physicians used, in part, to support their opinions. Consequently, Dr. Mann's opinion is not as well documented as the other medical opinions. Second, the other physicians, including board certified pulmonologists, included Dr. Mann's treatment notes in their review of the entire medical record. Because the other doctors were able to obtain Dr. Mann's observations and diagnoses, Dr. Mann was not in sole possession of unique medical information concerning Mr. Bridges' pulmonary condition.

Since Dr. Mann's medical opinion does not have greater probative significance, all the medical opinions on this issue have the same relative probative value. And, because all the opinions are equally probative, I am persuaded by the consensus of several physicians, rather than Dr. Mann's sole, contrary conclusion, that asthma was neither related to, nor substantially aggravated by, Mr. Bridges' exposure to coal dust. Accordingly, I find his asthma was not legal pneumoconiosis.

Emphysema

The question of whether Mr. Bridges' emphysema, or COPD, was related to, or aggravated by, his coal mine employment divided the medical opinion in this case. Again, my first step in the evaluation of the diverse medical opinions on this issue is to assign relative probative weight.

Of the 17 physicians who either provided medical treatment to Mr. Bridges, or evaluated the medical evidence in this case, several doctors did not specifically evaluate or discuss the etiology of Mr. Bridges' emphysema.⁴⁹ The remaining assessments relating to the cause of the emphysema are all generally well documented. However, as discussed below, some of the opinions were better reasoned and consequently more probative.

As Mr. Bridges' treating physician, Dr. Mann was in an excellent position to provide a well-reasoned opinion on the source of Mr. Bridges' emphysema. Unfortunately, in the referenced statement from February 1994, Dr. Mann did not clarify whether the occupational, or coal workers,' pneumoconiosis, he believed caused Mr. Bridges' respiratory failure included Mr. Bridges' emphysema or COPD. Even if I believed his statement showed a link between the emphysema and coal dust, the terse nature of the statement precludes my ability to determine the basis for his conclusion. The February 1994 statement is not well reasoned and has little probative value.

Dr. Modlin, as the attending physician at the time of Mr. Bridges' death was also situated to provide an insightful conclusion. However, his death certificate diagnosis of "black lung (COPD)" loses probative value for two reasons. First, based on his check mark on the death certificate that no autopsy had been accomplished, it appears Dr. Modlin was not aware of either Dr. Ahmed's autopsy report or Dr. Rodman's pathology findings. His diagnosis, apparently including COPD within his finding of black lung disease, is not as well documented as the other medical assessments that were based, in part, on the probative autopsy and pathology reports. Second, and more significant, since the record contains no explanation by Dr. Modlin on how he arrived at death certificate diagnosis, his opinion is not well reasoned.

Likewise, Dr. Daniel's conclusion that COPD was not caused by coal dust is not well reasoned. On the pulmonary examination report, Dr. Daniel failed to explain the basis for his determination that coal dust was not a factor in Mr. Bridges' struggle with COPD.

Dr. Rodman's conclusion that the emphysema was not related to Mr. Bridges' coal mine employment is well documented, reasoned, and probative. Under the microscope, Dr. Rodman found panacinar emphysema in Mr. Bridges' the lung tissue. Based on his observation, Dr. Rodman provided a highly descriptive statement that the coal residue within the diffuse and extensive emphysematic tissue, was due to, and not the cause of, the emphysema. He specifically excluded coal dust as a cause of the emphysema. Instead, the black pigment was a secondary consequence of the diffuse emphysema.

⁴⁹Dr. Ahmed, Dr. Previll, Dr. Leef, Dr. Lapp, Dr. Walker, Dr. Hayes, and Dr. Pushkin.

Along a similar line of reasoning, Dr. Kleinerman and Dr. Hutchins relied on Dr. Rodman's pathology observations concerning the panacinar emphysema to conclude Mr. Bridges' emphysema was caused by his exposure to cigarette smoke and not coal dust. Due to the depth of their analyses and reliance on the specific, probative pathology findings, their opinions have enhanced probative weight.

Dr. Caffrey also provided a reasoned explanation concerning his determination, based on the finding of panacinar emphysema, that coal dust did not cause the emphysema in Mr. Bridges' case. He clearly stated the emphysema, based on its type, was not caused by coal dust. At the same time, Dr. Caffrey did admit that if a cigarette smoking coal miner developed COPD, he would not be able to distinguish the source of the COPD. Because his comment concerning a cigarette-smoking coal miner's COPD was presented in response to a hypothetical question and not directly to Mr. Bridges' case, Dr. Caffrey's answer does not necessarily impeach his conclusion in the case of Mr. Bridges, with the specific pathology findings, that coal dust was not a factor. However, Dr. Caffrey did not clarify the seeming inconsistency between his two statements. Absent further clarification, I give Dr. Caffrey's opinion on the emphysema etiology diminished probative value.

Based on the characteristics of Mr. Bridges' emphysema, Dr. Castle found Mr. Bridges' emphysema was not caused by coal dust. At the same time, without explanation, he opined that coal dust did contribute to Mr. Bridge's COPD. Absent any further explanation about the relationship between Mr. Bridges' non-coal dust induced emphysema and his coal dust related COPD, Dr. Castle's opinion had diminished probative value. His conclusion is ambiguous and not well reasoned.

Dr. Koenig's opinion is relatively well documented and generally reasoned. However, on the issue of emphysema and its relation to coal dust exposure, his finding that coal dust is a "possible" cause of, or contributing factor to, Mr. Bridges' panacinar emphysema falls short in terms of relative probative weight. By indicating coal dust was a "possible" source or factor, Dr. Koenig has presented an ambiguous, or less than definitive, statement concerning the emphysema's etiology. More importantly, his analysis of the relationship between coal dust exposure and emphysema is not as well reasoned as other, more detailed, medical opinions in this case on the subject. Notably, Dr. Koenig did not discuss the different types of emphysema and how the type of emphysema may be related to the cause of the respiratory damage. Although Dr. Koenig asserts the presence of classic pneumoconiosis increases the likelihood that coal dust also played a role in the emphysema, his lack of thorough discussion about the emphysema, in light of Dr. Rodman's highly detailed pathology findings demonstrating the presence of only panacinar emphysema, lessens the probative value of his opinion.

Dr. Gaziano provided a well reasoned explanation concerning the different types of emphysema and their causes. Cigarette smoke typically produces diffuse central lobular emphysema while coal dust causes focal emphysema. Because the pathology report indicated the presence of only panacinar emphysema and not focal emphysema, Dr. Gaziano did not think coal dust produced the type of emphysema in Mr. Bridges' lungs. Consequently, I considered Dr. Gaziano's analysis supportive and probative of the position that coal dust did not cause Mr. Bridges' emphysema.

In summary, I did not find the less probative opinions of Dr. Mann, Dr. Modlin, Dr. Daniel, Dr. Caffrey, and Dr. Castle helpful in resolving this issue. Of the remaining probative medical assessments, Dr. Koenig's opinion that Mr. Bridges' emphysema is related to coal dust exposure is outweighed by the better reasoned, and pathology specific, medical opinions of Dr. Rodman, Dr. Kleinerman, Dr. Hutchins, and Dr. Gaziano. Based on this consensus of better reasoned medical opinion, I find Mr. Bridges' panacinar emphysema was caused solely by cigarette smoke and was not related to, nor aggravated by, coal dust exposure. The preponderance of the more probative medical opinion does not support a finding that Mr. Bridges' emphysema is legal pneumoconiosis.

Issue # 3 Pneumoconiosis Arising Out Of Coal Mine Employment.

While the medical evidence has established that Mr. Bridges had at least classic pneumoconiosis, Mrs. Bridges must also demonstrate that her husband's pneumoconiosis arose out of coal mine employment. As indicated earlier, under the regulations, if a miner works ten or more years in one or more mines, a presumption exists that his or her pneumoconiosis arose out of coal mine employment. Since the parties have stipulated Mr. Bridges worked at least 26 years as a coal miner, the presumption that Mr. Bridges' pneumoconiosis arose out of his coal mine employment exists, and there is insufficient evidence to rebut that presumption. Accordingly, Mrs. Bridges has established the third element of a survivor claim, pneumoconiosis arising out of coal mine employment.

Issue # 4 - Death Due to Pneumoconiosis

Having proved the first three elements of entitlement, Mrs. Bridges may receive survivor benefits if the preponderance of the evidence in the record establishes that her husband's death was due to pneumoconiosis. As previously discussed, the regulations provide four methods for showing death due to pneumoconiosis. Since there is insufficient evidence of complicated pneumoconiosis in this case, Mrs. Bridges may not invoke the regulatory presumption of causation. As a result, Mrs. Bridges must show Mr. Bridges's death was caused by pneumoconiosis, or his death was caused by complications of pneumoconiosis, or pneumoconiosis was a substantially contributing cause or factor leading to Mr. Bridges's death. Based on my findings that neither Mr. Bridges' asthma/asthmatic bronchitis nor his emphysema are legal pneumoconiosis, my focus in considering the three means to establish death due to pneumoconiosis will be on the classic pneumoconiosis established by Dr. Rodman's pathology report.

Death Caused By Pneumoconiosis

Once again, one physician has expressed an opinion that is opposite to the opinion of the remaining doctors evaluating Mr. Bridges' death. Dr. Modlin, who was the treating physician when Mr. Bridges passed away, indicated on the death certificate that "black lung (COPD)" was the cause of death. As the physician present at the time of death, Dr. Modlin had an opportunity to provide one of the most probative opinions on the cause of death. Yet, for the following reasons, I give his conclusion diminished probative value. First, due to the parenthetical expression "COPD," I am unable to discern whether Mr.

Modlin's reference to "black lung" referenced classic pneumoconiosis or coal dust-related COPD. The distinction is important because I have already determined that Mr. Bridges' obstructive pulmonary disease was not legal pneumoconiosis. Second, even if Dr. Modlin believed classic pneumoconiosis killed Mr. Bridges, and despite his attendance at Mr. Bridges' death, his opinion is not as well documented as other medical assessments because he did not reference either the autopsy report or the pathology findings. In fact, Mr. Modlin checked on the death certificate that no autopsy had been accomplished. He also annotated in the hospital record the absence of an autopsy. Third, Dr. Modlin's conclusion about the cause of death is not well-reasoned. He did not explain what factors led to his determination that black lung caused Mr. Bridges' death.

None of the other medical experts in this case believed pneumoconiosis caused Mr. Bridges to die. Instead, the near unanimous documented and reasoned medical conclusion is that cardiac arrhythmia was the immediate cause of Mr. Bridges' demise.⁵⁰ In addition, Dr. Lapp, Dr. Rodman, Dr. Walker, Dr. Hayes, Dr. Pushkin, Dr. Kleinerman, Dr. Hutchins, Dr. Caffrey and Dr. Castle specifically excluded pneumoconiosis as a direct cause of death. In light of this overwhelming consensus and considering the diminished probative value of Dr. Modlin's opinion, I find Mr. Bridges' death was not caused by pneumoconiosis.

Death Caused By Complications Of Pneumoconiosis

Numerous physicians considered whether complications stemming from the presence of pneumoconiosis were factors in Mr. Bridges' death.⁵¹ Dr. Mann found that Mr. Bridges' cardiac arrhythmia was brought on by respiratory failure primarily caused by pneumoconiosis. Since the cardiac arrhythmia and respiratory failure were complications of pneumoconiosis according to Dr. Mann, his assessment supports a finding that Mr. Bridges' death caused by complications associated with black lung disease.

Based on the pathology finding that 20% of Mr. Bridges' lung tissue contained pneumoconiosis and considering his respiratory distress just prior to his death, Dr. Gaziano opined that classic pneumoconiosis was a "significant" contributing factor in Mr. Bridges' death. Mr. Bridges' respiratory distress, a pulmonary health complication caused in part by pneumoconiosis, eventually led to the cardiac arrest. Consequently, a pneumoconiosis complication was involved in Mr. Bridges' death.

After finding myocardial infarction, rather than pneumoconiosis, as the cause of death, Dr. Lapp opined that coronary artery disease and emphysema were contributing factors in Mr. Bridges' death.

⁵⁰Dr. Mann, Dr. Rodman, Dr. Gaziano, Dr. Koenig, Dr. Caffrey, and Dr. Castle believed the actual cause of death was cardiac arrest or arrhythmia. Mr. Lapp opined Mr. Bridges suffered a myocardial infarction.

⁵¹The following physicians either did not address the issue or found some other complication was involved: Dr. Ahmed, Dr. Prevail, Dr. Leef, Dr. Daniel, and Dr. Modlin (myocardial failure was a contributing factor).

Dr. Rodman believed the principal contributing factors in this case were Mr. Bridges' asthma and cigarette smoke-related emphysema. And, while Dr. Rodman indicated pneumoconiosis may have been a minor "contributing factor," he found no connection between Mr. Bridges' heart arrhythmia and pneumoconiosis. As a result, Dr. Rodman's opinion supports a finding the death-producing heart arrhythmia was not a complication of pneumoconiosis.

Both Dr. Walker and Dr. Hayes, based on an additional review of the medical record, including the probative pathology report, concluded Mr. Bridges' pneumoconiosis was "insignificant" and played no role in his death. Dr. Pushkin agreed with their assessment.

Because Mr. Bridges' classic pneumoconiosis did not produce any "clinically significant" impairment, Dr. Koenig believed classic pneumoconiosis did not contribute significantly to his death.⁵² Instead, Dr. Koenig focused on COPD as the major respiratory illness causing the heart-stopping death. His opinion does not support a finding that a complication of classic pneumoconiosis was involved.

Although Dr. Kleinerman didn't know what actually generated Mr. Bridges' fatal heart arrhythmia, he found his breathing problems were not caused by simple pneumoconiosis and black lung disease did not play any role in the death.

Likewise, Dr. Caffrey concluded the coal workers' pneumoconiosis in 20% of Mr. Bridges' lungs was neither extensive nor severe. This "mild" case of classic pneumoconiosis played no significant role in his death because the amount of pneumoconiosis would not cause cardiac arrest.

Dr. Hutchins concluded Mr. Bridges' coal workers' pneumoconiosis was insufficient to contribute to, or cause, a respiratory impairment. He noted that the pneumoconiosis was not found in the majority of the x-ray interpretations and the pathology examination found only 20% of the lung tissue contained simple pneumoconiosis. Simple pneumoconiosis did not contribute to Mr. Bridges' death.

Because the chest x-rays failed to show the pneumoconiosis found during the microscopic pathology examination, Dr. Castle considered Mr. Bridges' coal workers' pneumoconiosis to be "mild." Considering the mild degree of black lung disease, it played no role in Mr. Bridges' death.

Of the documented and reasoned medical opinions, only Dr. Mann and Dr. Gaziano believed classic pneumoconiosis, by complicating Mr. Bridges' pulmonary condition, led to his death. The remaining ten physicians, based either on the absence of a connection between the heart arrhythmia and black lung disease or a determination that Mr. Bridges had only a mild case of coal workers' pneumoconiosis, concluded coal workers' pneumoconiosis was not a factor in his death. In particular, I found the opinions of Dr. Koenig, Dr. Caffrey, and Dr. Hutchins on the matter very well documented, reasoned and probative.

⁵²D. Koenig certainly believed Mr. Bridges' coal mine employment caused or contributed to Mr. Bridges' severe COPD which eventually led to his death. However, as previously mentioned, I have determined Mr. Bridges' COPD, or emphysema, was not pneumoconiosis.

The preponderance of the medical opinion does not support a finding that complications of pneumoconiosis caused Mr. Bridges' death.

Pneumoconiosis Was a Substantially Contributing Cause Of, Or Hastened, Death

On the issue of whether pneumoconiosis hastened death, nearly the same split in medical opinion occurred. Since Dr. Mann opined pneumoconiosis lies as the core illness which eventually caused respiratory failure and cardiac arrest, his opinion also supports the proposition the pneumoconiosis hastened Mr. Bridges' death. In other words, as the fundamental, underlying pulmonary problem which set the fatal chain reaction in motion, pneumoconiosis hastened Mr. Bridges' death.

Although Dr. Gaziano did not directly address whether Mr. Bridges' case of significant pneumoconiosis hastened his passing, he did believe Mr. Bridges' prolonged respiratory distress, in part stemming from pneumoconiosis, contributed to death. Based on that language, I consider Dr. Gaziano's opinion supportive of the determination that pneumoconiosis hastened death.

On the other hand, Dr. Lapp determined pneumoconiosis was not a factor in Mr. Bridges' death, so his opinion supports a finding that black lung disease did not hasten death in this case. Likewise, Dr. Walker and Dr. Hayes, with the support of Dr. Pushkin, also agreed that the insignificant pneumoconiosis did not hasten Mr. Bridges' death. And, since simple pneumoconiosis played no, or significant, role in Mr. Bridges' death, Dr. Kleinerman, Dr. Caffrey, and Dr. Hutchins, specifically stated it did not hasten his death.

Due to the absence of any connection between Mr. Bridges' coal workers' pneumoconiosis and his heart's irregular beat, Dr. Rodman concluded Mr. Bridges would have died at the same time and in the same way whether or not he had pneumoconiosis. As a result, Dr. Rodman obviously does not believe pneumoconiosis hastened death.

Dr. Koenig and Dr. Castle did not rank classic pneumoconiosis as a significant factor or state pneumoconiosis hastened Mr. Bridges' death.

Again, Dr. Mann and Dr. Gaziano's medical opinions are opposed by the substantial majority of the physicians in this case. I am persuaded both by the preponderance of the medical opinion and Dr. Rodman's well documented and reasoned medical opinion that coal workers' pneumoconiosis did not hasten Mr. Bridges' death.

CONCLUSION

Mrs. Bridges is an eligible survivor who has proven that her deceased husband, Mr. George H. Bridges, had coal workers' pneumoconiosis. However, the preponderance of the probative medical opinion in this case does not support a finding that pneumoconiosis or its complications caused, contributed

to, or hastened Mr. Bridges' death. Considering the whole record, I find Mrs. Bridges is not able to prove that Mr. Bridges' death was due to pneumoconiosis, the requisite last element of entitlement. Having failed to meet her burden of proof on the last element of entitlement, Mrs. Bridges' claim for survivor benefits must be denied.

ORDER

The claim for survivor benefits under the Act of Mrs. MONA T. BRIDGES is **DENIED**.

SO ORDERED:

RICHARD T. STANSELL-GAMM
Administrative Law Judge

Washington, D.C.

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. §725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date this decision is filed with the District Director, Office of Worker's Compensation Programs, by filing a notice of appeal with the Benefits Review Board, ATTN.: Clerk of the Board, Post Office Box 37601, Washington, DC 20013-7601. See 20 C.F.R. §725.478 and §725.479. A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits. His address is Frances Perkins Building, Room N-2117, 200 Constitution Avenue, NW, Washington, DC 20210.